

care and caution in these patients. The now known untoward effects should be anticipated or death may ensue.

Any scheme for management should include (1) prolonged gastric aspiration and lavage, as the drug is excreted into the stomach via the gastric secretions; (2) availability of immediate assisted ventilation; (3) immediate cardiac monitoring and availability of emergency equipment; (4) the intravenous use of potassium chloride and lidocaine as indicated; (5) control of seizures with paraldehyde or diazepam; and (6) keen awareness of the possible sudden deterioration of the patient.

PAUL TREITMAN, MD
Panorama City, Calif

1. Young JA, Galloway WH: Treatment of severe imipramine poisoning. *Arch Dis Child* 46:353-355, 1971.
2. Penny R: Imipramine hydrochloride poisoning in childhood. *Amer J Dis Child* 116:181-186, 1968.

The Gaisböck Syndrome

To the Editor.—The "Gaisböck Syndrome," although considered by some as a "nonentity," has the specific hematologic findings of an apparent polycythemia, a normal circulating red blood cell (RBC) mass, and a decreased plasma volume. The name "stress polycythemia" was given on the basis of clinical manifestations and evidence of reactions to psychic stress in many of these patients. Its incidence has been reported to be higher in psychiatric patients than in normal controls, and a clear temporal relationship between clinical manifestations of anxiety and stress and measured blood volume changes were documented in a case report. However, no physiologic or pathologic basis has been found.

In our study, which was designed to elicit the pathophysiology of this entity, two typical cases were studied. Both of the patients had hematocrit readings ranging from 54% to 61% for at least one year and blood volume studies via the double tagging technique showed a normal RBC mass and a decreased plasma volume. The rest of the results of their blood studies (hematologic and chemistry) were completely normal on at least three occasions. Psychiatric evaluation by a diagnostic interview, Minnesota Multiphasic Personality Inventory (MMPI), Zung Depression scale, and Taylor Manifest Anxiety scale were carried out and compared to a similar evaluation carried out 12 months prior to this study. The different physiological factors that may

have been responsible for the blood volume alteration in our two patients, namely, increase in plasma histamine, steroids, or epinephrine, or a decrease in antidiuretic hormone production, were studied. Plasma histamine, diurnal plasma cortisol, 24-hour urinary 17-hydroxycorticosteroids, 24-hour urinary metanephrines and normetanephrines, as well as a 24-hour water deprivation test, followed by a Pitressin test were all within normal limits. The sleep record of both patients for five consecutive nights showed significant decrease in both mean rapid eye movement (REM) time and percentage in all five nights, though more so during the first two nights. A 12-hour (day-night) intake-output schedule showed a statistically higher daytime intake volume in both patients. Although there was no significant difference between daytime and nighttime urine output volume with the first patient, urine output volume was significantly higher during the night with the second patient. Nocturnal urine in patient 1 had essentially the same osmolality and specific gravity as that of daytime urine. In patient 2 both specific gravity and osmolality of nocturnal urine were significantly lower than those of daytime urine.

The psychiatric and psychometric findings in both patients were essentially similar to those observed 12 months prior to this study. Both patients exhibited symptoms and psychometric criteria indicative of their reacting to stress. Although their personalities were quite different, they did share traits of passivity, obsessionism, and tendency to somatization. The findings of decreased nocturnal water conservation and urine concentration are consistent with a decrease in nocturnal antidiuretic hormone (ADH)-like activity despite a grossly normal ADH. Rapid eye movement sleep mean time and percentage was found to be decreased in both patients, though more so in the first two nights due to "first night effect." Despite an expected REM rebound in the latter three nights, there was still significant decrease in REM time and percentage in these nights. Rapid eye movement sleep has been noted to decrease with psychic stress, though inconsistently so due to the "REM rebound" phenomenon. Increase in ADH-like activity has also been noted during REM sleep. Although patients who are chronically deprived of REM sleep have not been reported to develop this syndrome, our findings indicate that the stress relationship to

the blood volume change might be mediated via a primary decrease in REM sleep secondary to stress with subsequent decrease in nocturnal primary ADH-like activity and eventual nocturnal water loss. It seems that the rather mild blood volume change and essentially no plasma osmolality change seen in these patients fail to trigger the ADH feedback mechanism and a chronic mild and nonprogressive (due to REM-rebound) polycythemia persists in these predisposed patients.

M. KHALED EL-YOUSEF, MD
Tennessee Neuropsychiatric Institute
Nashville
WILLIAM E. BAKEWELL, Jr.
University of North Carolina
School of Medicine
Chapel Hill

Infectious Mononucleosis in Athletes

To the Editor.—Any discussion of the etiology of infectious mononucleosis (219:897, 1972) must give some consideration to its almost epidemic occurrence in athletes who have been trained excessively, and where particular coaches are known for their strenuous training programs.

This does suggest that infectious mononucleosis is related to stress and may indeed be another autoimmune disease. While mononucleosis is not supposed to recur, frank recurrences are fairly frequent and many patients report symptoms similar to the original attack while under stress conditions. The signs and symptoms of "staleness" in an athlete, fatigue, rash, swollen glands, joint pains, poor attention span, sleep disturbances and loss of appetite, parallel remarkably those of mononucleosis.

In my experience, many distance runners clearly relate overtraining to development of either respiratory or mononucleosis-like symptoms.

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Psychosis, Psychiatry, LSD 2256 a And Homicide

To the Editor.—The report "Homicide during a psychosis induced by LSD" (219:800, 1972) presents the "case history" of a 22-year-old graduate student who, while in Israel and while allegedly under the influence of LSD, killed one person and injured two others. A year before, after assaulting an elderly woman, "The police caught him and took him to a mental hospital where he recovered rapidly and was released in a few days." After the murder, "His parents had brought

him home from Israel after he had been declared unfit to stand trial for homicide because of insanity. On admission he was noted to be a lithe, handsome young man with a guarded manner but no evidence of psychosis." Certainly, the Israeli authorities could have waited till this killer's alleged psychosis cleared, and could have then tried and punished him.

Here, then, is the story of a young man who, between the ages of 21 and 22, was apprehended for one assault and one murder plus assault, but was not held legally responsible for any of these acts. Why not? Because under the guise of "mental illness," the medical profession provides society a psychiatric alibi for murder and mayhem.

It is a sign of our times that the depredations of a young man such as described in this case report should be published in a medical journal as if he were a "patient" and as if his crime would constitute a "medical" problem. Moreover, this case report is only a drop in the ocean: our current medical and psychiatric literature overflows with the "case histories" of such "patients." Truly, physicians and laymen alike have managed to convince themselves that bad is mad, and mad is sick—a conviction that loses none of its absurdity with its growing popularity. If the American medical profession continues to accept, and indeed to embrace, psychiatry as "just another" medical specialty—when it is, in fact, used as a political creed whose implacable hostility to the Anglo-American tradition of the Rule of Law is displayed ever more brazenly, as "case reports" such as this one and such as those daily emanating from Russia show—it will have only itself to blame for its own destruction.

THOMAS S. SZASZ, MD
State University Hospital
Upstate Medical Center
Syracuse, NY

To the Editor.—I must compliment Drs. Reich and Hepps for their excellent description of the literature on lysergic acid diethylamide (LSD). The fact, however, that the act was committed while a patient was under the influence of an intoxicant, be it an alcoholic or a psychoactive chemical, does not of itself establish a cause and effect relationship. We have seen cases where serious social and legal problems have occurred during drug intoxication. As the author so adequately points out, these "have become relatively common medical

emergencies."

My purpose is to indicate that it is unscientific, I think, and rather subjective to relate an incident which occurs during a cycle of a person's life, such as marital situation or job situation, to the particular drug that is involved. I do compliment the authors for their final paragraph.

The need for prompt and adequate treatment of adverse reactions "to any deleterious nostrum" is a very serious medical concern.

MYRON H. MARSHALL, MD
Buffalo

To the Editor.—The authors wonder at the phenomenon of two "bad trips" occurring in an individual who had several other pleasant experiences with lysergic acid diethylamide (LSD). I have reread the article and fail to find any clue as to how the authors know that the drugs involved were LSD. In fact, they liken the bad trips to those seen in amphetamine users, and it is well known that what is sold in the drug culture as LSD may be something else entirely or at least a combination of drugs, commonly including amphetamine. Without such proof as to the exact nature of the drugs taken, the report loses validity as a case report of a specific drug reaction.

GEORGE S. LAYNE, MD
PHS Outpatient Clinic
Buffalo

Alcohol in Breath, Blood, And Cerebrospinal Fluid

To the Editor.—Several studies indicate that the alcohol content of cerebrospinal fluid (CSF) roughly parallels that of blood.^{1,2} It is not known whether cellular and chemical constituents of spinal fluid are affected by alcohol. The present study had two goals, to compare breath, blood, and CSF alcohol concentrations in severely intoxicated alcoholics; and secondly to ascertain whether CSF constituents were abnormal when high levels of alcohol were present.

Methods.—Ten male alcoholics between the ages of 25 and 50 were admitted to an alcoholism inpatient service with blood alcohol levels, as determined by breath analysis, exceeding 150 mg/100 ml. Blood and lumbar spinal fluid samples were obtained immediately after the determination of breath alcohol. The blood and CSF were tested for alcohol, uric acid, sodium, chloride, potassium, magnesium, and glucose. The CSF additionally was tested for total protein, cells, and specific gravity, and the blood was tested for calcium. Gas liquid

chromatography was used in the determination of alcohol in breath, blood, and spinal fluid.

Results.—With the exception of potassium, which was slightly elevated in both blood and CSF, all values were within the normal range. No cells were observed in the CSF of seven subjects. In the other three, cell counts of 9, 5, and 3, respectively, were reported. Specific gravities were within normal limits. In a few instances, patients showed moderate elevations of one or more blood or CSF constituents. However, the elevations were not correlated with alcohol concentration or clinical manifestations; nor did deviations from normal occur simultaneously in blood and spinal fluid, suggesting they may have represented laboratory error.

Alcohol concentrations in breath, blood, and spinal fluid were significantly correlated but there were wide variations in individual instances (Table). There was no indication that

Comparison of Alcohol in Breath, Blood, and Spinal Fluid (mg/100ml)

Subject	Breath	Blood	Spinal Fluid
1	213	284	294
2	348	330	338
3	171	175	164
4	267	252	268
5	233	244	276
6	155	183	179
7	135	84	99
8	196	173	194
9	196	224	244
10	356	232	236

alcohol concentrations were consistently higher or lower in breath, blood, or spinal fluid, and the differences may represent variability inherent in the assay procedures rather than real differences in concentration. The fact that, in four instances, the breath analysis showed higher values than did the blood determinations has medico-legal implications with regard to the validity of extrapolating blood alcohol concentrations from breath analysis.

In view of a previous study³ in which an association was reported between hypomagnesemia and delirium tremens, it was interesting that magnesium levels in blood and spinal fluid were within normal limits in this group of severely intoxicated individuals. However, the two studies are not truly comparable, since our subjects were acutely intoxicated rather than withdrawing from alcohol.